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Family history and perceived risk of diabetes, cardiovascular disease, cancer and depression

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Abstract

Background: Family history is a useful and inexpensive tool to assess risks of multifactorial diseases. Family history enables individualized disease prevention, but its effects on perceived risks of various diseases need to be understood in more detail. We examined how family history relates to perceived risk of diabetes mellitus, cardiovascular disease (CVD), cancer, and depression, and whether these associations are independent of or moderated by sociodemographic factors, health behavior/weight status (smoking, alcohol consumption, physical activity, BMI [kg/m²]), or depressive symptoms.

Methods: Participants were Finnish 25−74-year-olds (N=6258) from a population-based FINRISK 2007 study. Perceived absolute lifetime risks (1–5) and first-degree family history of CVD, diabetes, cancer and depression, and health behaviors were self-reported. Weight and height were measured in a health examination.

Results: Family history was most prevalent for cancer (36.7 %), least for depression (19.6 %). Perceived risk mean was highest for CVD (2.8), lowest for depression (2.0). Association between family history and perceived risk was strongest for diabetes (β=0.34, P<0.001), weakest for depression (β=0.19, P<0.001). Adjusting for sociodemographics, health behavior, and depressive symptoms did not change these associations. The association between family history and perceived risk tended to be stronger among younger than among older adults, but similar regardless of health behaviors or depressive symptoms.

Discussion: Association between family history and perceived risk varies across diseases. People's current understandings on heritability need to be acknowledged in risk communication practices. Future research should seek to identify effective strategies to combine familial and genetic risk communication in disease prevention.

Keywords: family history; perceived risk; cardiovascular disease; diabetes; cancer; depression

1. Introduction

The general public is frequently reminded of health risks of certain lifestyle choices – such as smoking, unhealthy diet and lack of exercise – by mass media and health care professionals. The aim of risk communication is to increase risk perception and motivate preventive behavior (1). Several health behavior theories, including the Health Belief Model (2), assume perceived risk to be a key motivator of preventive behavior. In addition, people supposedly adjust their risk perceptions according to their current behavioral and other risk factors (3).

A form of risk information indicating inherited risk is family history, which is a useful and inexpensive tool to assess individual risks of multifactorial diseases (4,5). Nordic twin studies suggest heritability to be 20 % for type 2 diabetes (6) and 18–33 % for cancer (7,8), whereas heritability of depression appears to be 37−50 % (9,10). Early onset indicates familiality of cardiovascular diseases (CVD) (11), diabetes (12), cancer (13), and depression (9). Family history can serve as the cornerstone for individualized disease prevention, but its effects on perceived risks of various diseases need to be understood in more detail.

Most people understand genetics in terms of traits and diseases 'running in the family', instead of in terms of the structural and functional nature of genes (14,15). Family history has shown to be strongly linked to personal risk perceptions of CVD, type 2 diabetes and cancers (16–19), but to our knowledge, no previous study has explicitly compared this association across different diseases in the general population. Some diseases may be perceived more threatening than others, therefore perceived and actual risks could differ. Awareness of a familial risk may increase sense of self-control (20) and motivate preventive action, such as seeking information, attending screenings or attempting lifestyle changes (21). However, if family history leads to greater perceived risk for some diseases than others, preventive motivation may vary accordingly.

Research on people's understanding on etiology suggests that people know that familial diseases may be caused by both genetics and/or shared health behavior (14). Those who acknowledge the influence of genetics are also more aware of the role of lifestyle (22). There is evidence that most people consider genetic and behavioral causes of multifactorial diseases separately, one adding to the other (23). That is, most people ignore the interactive nature of genes and behavior. However, it

is unknown whether this is reflected in personal risk perceptions, for example, whether family history elevates smokers' risk perceptions to the same degree as non-smokers'.

In addition to actual risk factors like family history and health behavior, risk perceptions may reflect cognitive tendencies. DiLorenzo et al. (16) found perceived risks of different diseases to be interrelated. Personality traits (e.g. neuroticism) or depressive symptoms may partly explain this. Depressive symptoms may cause a pessimistic bias and thus increase risk perceptions. Moreover, this bias might contribute to genetic fatalism; depressive symptoms might amplify the association between family history and perceived risk.

The aim of this study was to examine whether family history was related to perceived risk of CVD, diabetes, cancer, and depression, and whether this association varied across diseases in the Finnish population. Furthermore, we explored whether associations between family history and perceived risk were modified by respondent's own health behavior/weight status (smoking, alcohol consumption, physical activity, body mass index [BMI kg/m^2]) or sociodemographics (gender, age, education). Finally, we examined whether respondent's current depressive symptoms were related to perceived risks of CVD, diabetes and cancer, and whether depressive symptoms moderated the relationships between family history and perceived risks of these diseases. The study extends previous literature by using a large population-based sample that enables exploration of several different possible modifiers of the association between family history and perceived risk.

2. Methods

2.1. Participants

The participants were 25–74-year-old Finnish men and women from the National FINRISK 2007 Survey conducted in January–March 2007 (24). A random sample of 10'000 people, stratified by gender and ten-year age groups was derived from a population register in five geographical regions (N=6258, response rate: 63 %). The participants got a mailed invitation to a health examination and a questionnaire on medical history, health behavior, and family history and personal risk perceptions of CVD, diabetes, cancer and severe depression. They filled it in at home and returned it at the municipal health center where they attended the health examination. Research protocols were designed and conducted in accordance with the guidelines of the Declaration of Helsinki, and approved by Ethics Committee of the Hospital District of Helsinki and Uusimaa. All participants

gave their written informed consent.

For analyses concerning depressive symptoms we used a sub-sample of the same participants who attended the Dietary, Lifestyle and Genetic determinants of Obesity and Metabolic syndrome (DILGOM) sub-study in April−June 2007 (N=5024, response rate: 80 %) (25). All the FINRISK 2007 participants were invited to this second study phase, which contained a health examination and various questionnaires, including one on depressive symptoms (26).

2.2. Measures

Perceived lifetime risks of CVD, diabetes, cancer, and severe depression were measured with single items: *How do you perceive your own risk of developing [disease] in your lifetime?* 0=*I have [disease]*, 1=*very low*, 2=*low*, 3=*moderate*, 4=*high*, 5=*very high*. Those who reported currently having CVD (N=292), diabetes (N=191), severe depression (N=61), or having/having had cancer (N=184) were excluded from analyses concerning that disease. In a recent study, a similar five-point scale correlated highly with a more continuous measure of perceived absolute risk, and moderately with perceived comparative risk (27).

Family history of CVD was assessed with questions on whether a) father, b) mother, c) one or more brothers d) one or more sisters of the participant had encountered a myocardial infarction prior the age of 60 (in case of mother 65). Family histories of diabetes, cancer and depression were assessed with items on whether a) father, b) mother, c) one or more brothers, d) one or more sisters of the participant had been diagnosed with the disease. These items were summed to form a 'family history' variable (scale 0−4) for each disease.

Age group comparisons were made between younger adults (25−39 years, 25.1 %), middle-aged (40−59, 42.6 %) and older adults (60−74, 32.3 %).

Education years were measured with a single item: '*How many years have you attended school or studied full time altogether?*' For group comparisons, education years were divided into tertiles (indicating low, middle and high level of education) according to birth year.

Smoking was divided into three categories, 1=never smokers (53.9 %), 2=former smokers (25.3 %) and 3=current smokers (20.3 %). Current smokers reported smoking regularly more than once a day for at least one year, including the preceding month.

Average weekly alcohol consumption (grams of pure alcohol per week) during the last 12 months was measured and calculated by asking respondents to describe their usual frequency and quantity of consuming different alcoholic beverages (28). To reduce skewness (4.55) and kurtosis (28.07) of the distribution we used square root transformation before conducting correlative and regression analyses. Group comparisons were made by tertiles.

Leisure time physical activity was assessed with a single item, which has shown good criterion validity against morbidity and mortality and moderate correlation against accelerometer counts among the working age population (29): '*How much do you exercise and strain yourself physically in your free time?*' Response choices were 1=reading, television or physically non-exhausting work at home (sedentary, 20.3 %), 2=walking, cycling or similar at least 4 h/week excluding travel to work (moderately active, 53.2 %), 3=vigorous exercise or work at least 3 h/week and 4=competitive training of strenuous sports several times a week. Since few participants (N=97) responded 4, we combined 3 and 4 for group comparisons (active, 26.0 %).

BMI was calculated as weight (kg) divided by squared height (m), which were measured by trained research nurses in the health examination. For group comparisons, categories 1=normal weight (BMI=18.50−24.99, 36.0 %), 2=overweight (BMI=25.00−29.99, 40.4 %) and 3=obese (BMI≥30.00, 22.8 %) were created (underweight participants were excluded from group comparisons, N=37), based on the World Health Organization classification (30). Even though BMI is no actual measure of behavior, for simplicity, we refer to BMI, physical activity, alcohol consumption, and smoking by 'health behaviors'.

Depressive symptoms were measured with the Center for Epidemiological Studies Depression Scale (26) in the DILGOM sub-study.

2.3. Statistical analyses

Descriptive statistics were used to explore means, standard deviations, and distributions of family history and perceived risk of CVD, diabetes, cancer, and depression; sociodemographics (gender, age, education); health behaviors (smoking, alcohol consumption, BMI, physical activity); and depressive symptoms. Gender differences were tested with analysis of variance (continuous variables) or chi-square test (categorical variables). We used Pearson's correlation coefficients to examine bivariate associations between the studied variables, and a calculation for the test of the difference between two independent correlation coefficients (31,32) to test whether the correlation between family history and perceived risk varied statistically significantly across diseases.

We performed multivariate regression analyses − separately for each disease − to examine how perceived risk can be explained by family history, sociodemographics, health behaviors and depressive symptoms. Family history of the disease was included in the first step. Sociodemographics were added in the second, health behaviors in the third, and depressive symptoms in the fourth step. In the correlative and regression analyses, all variables were used as continuous, except for gender (0=men, 1=women) and smoking (0=never smokers/former smokers, 1=current smokers).

Interactions between family history and sociodemographics/health behaviors/depressive symptoms in relation to perceived risk were tested separately using models in which the interaction term was added to the model after the respective main effects (unadjusted analyses). In case of education also age was added in the first step, to control for increase in the overall education level during the past decades in Finland. In the interaction analyses, three-category smoking (never smokers as a reference category) and three-category physical activity (sedentary group as a reference category) variables were used.

3. Results

Table 1 presents descriptive characteristics of the sample. Perceived risk means were between 2 (low) and 3 (moderate); highest for CVD (mean=2.8, SD=0.9), lowest for depression (mean=2.0, SD=0.9). Of the participants, 25.3 % reported at least one family member with a myocardial

infarction prior the age of 60 (in case of mother 65). At least one family member with diabetes was reported by 28.4 %, with cancer by 36.7 %, and with depression by 19.6 % of the participants.

Table 2 shows Pearson's correlation coefficients between the studied variables. The test of the difference between two independent correlations indicated that family history-perceived risk correlation (r=0.33, P<0.001) was stronger for diabetes than for other diseases (P<0.001). Between CVD (r=0.26, P<0.001) and cancer (r=0.23, P<0.001) there was no statistically significant difference (P=0.107) in the respective correlations. For depression, the family history-perceived risk correlation (r=0.19, P<0.001) was weaker than for cancer (P=0.034), CVD (P<0.001), or diabetes (P<0.001).

Perceived risks of the four diseases correlated moderately with each other: strongest between CVD and diabetes (r=0.43, P<0.001), next between CVD and cancer (r=0.35, P<0.001). Family histories, however, correlated weakly with each other: strongest between CVD and diabetes (r=0.17, P<0.001), next between diabetes and depression (r=0.12, P<0.001). Those with family CVD history perceived their diabetes risk slightly higher than others (r=0.11, P<0.001), and vice versa (r=0.09, P<0.001). Those with higher BMI perceived their risks of diabetes (r=0.34, P<0.001) and CVD (r=0.25, P<0.001) considerably higher than others. Physically active participants perceived their risks lower than the less active. Current smokers perceived their risk of cancer and CVD higher than others, whereas alcohol consumption was only weakly related to perceived risks.

Table 3 summarizes the results of multivariate regression analyses on perceived risks. In step 1, family history was related to perceived risk for each disease. Adding sociodemographics (step 2) or health behaviors (step 3) to the regression models did not change these relationships. Using the DILGOM sub-sample, we further explored the effect of depressive symptoms on perceived risks of CVD, diabetes, and cancer. Adding depressive symptoms (step 4) to the regression models did not change the relationship between family history and perceived risk for any of the three diseases, even though depressive symptoms were related to perceived risk of CVD (β=0.13, P<0.001), diabetes (β=0.16, P<0.001), and cancer (β=0.14, P<0.001).

3.1. Interactions between health behavior and family history

We examined whether the relationship between family history and perceived risk was similar among those with healthy and unhealthy behaviors. As shown in Table 4, three out of the 16 tested interactions were statistically significant ($P<0.05$). To interpret these findings, we analyzed family history-perceived risk associations separately in groups: Unadjusted association between family history and perceived CVD risk was slightly weaker among former smokers (B=0.31, $P<0.001$) compared to never smokers (B=0.44, $P<0.001$) and current smokers (B=0.41, $P<0.001$). The association between family history and perceived depression risk was stronger among the normal weight (B=0.37, P<0.001) compared to the overweight (B=0.27, P<0.001) and obese (B=0.26, $P<0.001$). Also, it was stronger among those who consumed less alcohol (lowest tertile B=0.37, second tertile B=0.29, highest tertile B=0.28, all Ps<0.001). No interactions between physical activity and family history were found in relation to perceived risk.

Six out of the 12 tested interactions between sociodemographics and family history in relation to perceived risk were statistically significant ($P<0.05$, Table 4). Unadjusted associations between family history and perceived risk of CVD, diabetes and cancer were stronger among younger than among older adults, clearest for cancer (younger adults B=0.49, middle-aged B=0.31, older adults B=0.25, all Ps<0.001). Association between family history and perceived depression risk was stronger among women (B=0.35, $P<0.001$) than men (B=0.23, $P<0.001$). Associations between family history and perceived risk of CVD (low education B=0.33, middle education B=0.40, high education B=0.48, all Ps<0.001) and diabetes were slightly stronger among the highly educated than the less educated. No interactions between depressive symptoms and family history were observed in relation to perceived risk of CVD, diabetes or cancer.

4. Discussion

Family history was related to perceived risk of diabetes, CVD, cancer and depression independently of health behavior, sociodemographics, and depressive symptoms. The association was strongest for diabetes and weakest for depression. Among younger adults, the relationship between family history and perceived risk tended to be slightly stronger compared to older adults. We found no systematic differences between those with healthy and unhealthy behavior, or people with different levels of depressive symptoms, with regard to how their family history contributed to their perceived risks.

The finding that the association between family history and perceived risk was somewhat stronger for diabetes compared to the other studied diseases might indicate that people think diabetes is more heritable. In an earlier Finnish survey, diabetes (48%) and CVD (43%) were most commonly mentioned hereditary diseases, whereas only a fifth mentioned cancer or psychiatric diseases (15). Media publicity around a nationwide diabetes prevention program (33) that included screening for family history may have influenced these impressions. For depression, the association between family history and perceived risk was slightly weaker compared to somatic diseases. This might indicate low awareness of its heritability (34). However, family history questions concerned diagnosed diseases, which could have weakened the association between family history and perceived depression risk. Respondents might not only be aware of diagnosed depression, but also current or previous depressive symptoms of their family members, which could similarly increase their own risk perception. Research is needed on whether raising awareness of heritability of depression could be a way to encourage early help seeking.

Among younger and more educated participants, family history was slightly more strongly linked to perceived risks. This may be partly because younger participants' ill family members are also likely to be relatively young, and people might know that early onset indicates heritability. Also, younger and more educated people know more about genetics (15). Among women, family depression history was related to perceived risk more strongly than among men, even though heritability of depression appears to be similar for both genders (10). Despite that depression is considerably more prevalent among females (35), males with a family history of depression should be recognized as a particularly vulnerable group neither recognizing their risk nor seeking help (36).

We observed a few statistically significant interactions between family history and smoking, alcohol consumption and BMI in relation to perceived risks. However, possibility of Type 1 error related to multiple testing needs to be considered when interpreting these findings, particularly since the interactions did not systematically concern any specific disease or any specific health behavior. Overall, it seems that there were no differences between those with healthy and unhealthy behavior, with regard to how their family history contributed to their perceived risks. These results are in line with previous research suggesting that, in general, people process familial and behavioral risk factors separately, one adding to the other (14). As scientific understanding on gene-behavior interactions increases (37), careful consideration is needed on how to communicate this complex information. Also, it is worth noting that some health behaviors were not clearly related to

perceived risks. This may be explained by optimistic bias (38) and complex bi-directionality between risk perceptions and preventive health behaviors (3,39). Those with risky behavior perceive higher risk, and those who perceive high risk adopt health behaviors to reduce it, but these patterns cannot be tackled in cross-sectional designs.

As in previous research (16), perceived risks of different diseases were interrelated, even though family histories of different diseases were not strongly related. Those with more depressive symptoms perceived all their disease risks higher than those with less symptoms. They may be more pessimistic in general, or more specifically regarding their own self-efficacy to change health behavior (40). However, interrelatedness of perceived risks of CVD, diabetes and cancer remained highly similar after adjusting for depressive symptoms or health behaviors (results not shown). Neither did depressive symptoms amplify the relationship between family history and perceived risk of CVD, diabetes, or cancer; depressed people perceived their family history no more fatalistic than other people. Hence, depressive symptoms may manifest in more pessimistic risk perceptions, but interrelatedness of perceived risks needs to be studied further. Furthermore, although family history of one disease has been hypothesized and observed to be related to lower perceived risks of other diseases (16,41), we found no such pattern. Family history of one disease was mostly unrelated to perceived risks of other diseases. Yet, those with family diabetes history perceived their CVD risk *higher* than others, and vice versa (Table 2). Many people are likely to be aware of shared risk factors and comorbidity of diabetes and CVD, since these are stated in the national Current Care Guidelines (42). Per se, family history of one disease seems not to distract risk perceptions of other diseases.

Personalized medicine aims to customize risk information, for example by providing information on genetic risks (39). Family history provides social context and baseline for genetic risk communication. Shiloh et al. (43) found that genetic test results indicating diabetes risk increased perceived risk only among those who had a family history of diabetes. People's current understandings on hereditary mechanisms of illnesses need to be taken into account when communicating genetic risks (43,44). It is a challenge to present genetic risk information of multifactorial diseases in a way that evokes preventive motivation instead of fatalism, since prevention requires effortful lifestyle change and maintenance. Despite concerns, raising awareness of family history seems to increase sense of self-control (20) and preventive motivation (21). However, preventive motivation depends not only on risk perceptions but also on perceived severity and controllability (45), which vary across diseases (46). Future research should seek to identify the

most effective strategies to deliver familial and genetic risk information of different multifactorial diseases in a way that results in relevant health behavior change.

4.1. Study limitations and strengths

Strengths of this study included being able to study family history and perceived risk of several common diseases – posing considerable challenges for public health – at the same time. The large population-based health survey was most likely less biased towards those particularly interested in heritability, compared to studies focusing explicitly on inherited risks. However, participants were probably more aware of risk factors than the average population, since those with a lower education and a higher risk of mortality are more likely to opt out of the FINRISK study (47).

Limitations concern some of the measures. The studied disease categories were heterogeneous; different types of diabetes, cancer or CVD were not specified. Accuracy of self-reported family history is highest for first-degree relatives, but varies across diseases (48). We treated family history variables as continuous, but there were few people reporting more than two family members having the same disease. Family CVD history was measured narrowly (concerning only early myocardial infarctions) and all family history measures ignored possibility of several affected siblings. However, parental history was related to perceived risk very similarly as family history (results not shown). No conclusions on causalities can be made based on this cross-sectional study, even though increased risk perception most probably follows family history, not vice versa.

5. Conclusion

Family history was related to perceived risk strongest for diabetes, next for CVD and cancer, and weakest for depression, independently of sociodemographics, health behavior, and depressive symptoms. Moreover, our findings suggest that family history contributes to perceived risk similarly among people with healthy and unhealthy behaviors. Future research should explore how to combine family history and genetic risk communication in disease prevention.

Table 1
Descriptive characteristics of the FINRISK 2007 study sample.

	Women (N= 3099–3324)	**Men** (N=2645–2934)	**Total** (N=5744–6258)	**Range**
Age mean (sd)	50.2 (14.0)***	51.4 (13.9)	50.8 (13.9)	25.0−74.0
Education years mean (sd)	13.1 (4.0)***	12.3 (3.9)	12.8 (4.0)	0.0−50.0
Alcohol (g/week) mean (sd)	42.4 (88.5)***	114.6 (177.1)	76.4 (142.2)	0.0−1590.0
BMI (kg/m2) mean (sd)	26.9 (5.4)***	27.4 (4.2)	27.1 (4.9)	16.0−63.3
Normal weight, %	42.2***	29.1	36.0	
Overweight, %	33.2***	48.5	40.4	
Obese, %	23.4	22.1	22.8	
Smoking				
Never smokers, %	63.5***	43.1	53.9	
Former smokers, %	19.3***	32.2	25.3	
Current smokers, %	16.9***	24.1	20.3	
Physical activity				
Sedentary, %	19.7	21.1	20.3	
Moderately active, %	55.1**	51.1	53.2	
Active, %	24.7*	27.4	26.0	
Family history ≥ 1, %				
CVD	26.4	24.1	25.3	
T2D	30.7***	25.8	28.4	
Cancer	37.9	35.2	36.7	
Depression	22.9***	16.0	19.6	
Perceived risk mean (sd)				
CVD	2.8 (0.9)	2.8 (0.9)	2.8 (0.9)	1.0−5.0
T2D	2.5 (0.9)***	2.4 (0.9)	2.4 (0.9)	1.0−5.0
Cancer	2.7 (0.8)***	2.6 (0.8)	2.7 (0.8)	1.0−5.0
Depression	2.1 (0.9)***	2.0 (0.9)	2.0 (0.9)	1.0−5.0
Depressive symptoms[a] mean (sd)	10.6 (7.8)***	9.7 (7.2)	10.2 (7.5)	0.0−51.0

[a] subsample N=4913

*** gender difference $P \leq 0.001$ (ANOVA/Chi-Square).
** $P<0.01$.
* $P<0.05$.

Table 2
Pearson's correlation coefficients between the studied variables.

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1 perceived risk CVD															
2 perceived risk diabetes	.43***														
3 perceived risk cancer	.35***	.28***													
4 perceived risk depression	.24***	.30***	.29***												
5 family history CVD	.26***	.09***	.00	.04*											
6 family history diabetes	.11***	.33***	-.01	.02	.17***										
7 family history cancer	.01	.01	.23***	-.01	.03*	.06***									
8 family history depression	.04**	.04*	.02	.19***	.04**	.12***	.02								
9 gender men=0, women=1	.01	.06***	.07***	.09***	.03	.04**	.01	.08***							
10 age	.07***	.05**	-.02	-.07***	.20***	.15***	.31***	-.02	-.05**						
11 education	-.05***	-.05***	.04*	.03*	-.16***	-.14***	-.16***	.02	.11***	-.46***					
12 smoking 0=no, 1=yes	.09***	-.01	.14***	.05***	-.02	.00	-.07***	.01	-.09***	-.14***	-.07***				
13 alcohol consumption	.05***	-.02	.06***	.03*	-.04*	-.04*	-.04*	-.01	-.33***	-.09***	.06***	.24***			
14 BMI	.25***	.34***	.03*	.00	.09***	.10***	.09***	-.03*	-.06***	.20***	-.17***	.01	.03*		
15 physical activity	-.16***	-.15***	-.06***	-.11***	-.04**	-.05***	-.03*	.00	-.03*	-.08***	.14***	-.13***	-.02	-.22***	
16 depressive symptoms[a]	.17***	.20***	.15***	.48***	.07***	.04*	.05**	.10***	.08***	.10***	-.12***	.06***	.03	.10***	-.18***

***P<0.001 (2-tailed).
**P<0.01 (2-tailed).
*P<0.05 (2-tailed).
Cases excluded listwise (N=5113).
[a] subsample (N=4046)

Table 3
Results from multivariate regression analyses predicting perceived risk (FINRISK 2007).

		CVD(N=5445)				Diabetes(N=5529)				Cancer(N=5543)				Depression(N=5635)			
		B	SE B	β	Adj. R^2	B	SE B	β	Adj. R^2	B	SE B	β	Adj. R^2	B	SE B	β	Adj. R^2
Step 1					.07				.12				.05				.04
	Family history	.41	.02	.26***		.49	.02	.34***		.27	.02	.23***		.31	.02	.19***	
Step 2					.07				0.12				.07				.05
	Family history	.41	.02	.26***		.49	.02	.34***		.31	.02	.26***		.30	.02	.19***	
	Gender	.01	.02	.01		.09	.02	.05***		.10	.02	.06***		.14	.02	.07***	
	Age	.00	.00	.01		-.00	.00	-.02		-.01	.00	-.10***		-.01	.00	-.07***	
	Education	-.00	.00	-.01		-.00	.00	-.02		.01	.00	.02		-.00	.00	-.01	
Step 3					.15				.22				.10				.06
	Family history	.39	.02	.25***		.45	.02	.32***		.31	.02	.27***		.30	.02	.19***	
	Gender[a]	.05	.02	.03*		.10	.02	.06***		.15	.02	.09***		.16	.03	.09***	
	Age	-.00	.00	-.00		-.01	.00	-.07***		-.00	.00	-.07***		-.00	.00	-.07***	
	Education	.01	.00	.04***		.01	.00	.02		.01	.00	.05***		.00	.00	.00	
	Smoking[b]	.20	.03	.09***		-.02	.03	-.01		.29	.03	.14***		.06	.03	.03	

Alcohol	.01	.00	.04**	-.00	.00	-.00	.01	.00	.05***	.01	.00	.05***
BMI	.04	.00	.22***	.06	.00	.31***	.01	.00	.03*	.00	.00	.01
Physical activity	-.12	.02	-.10***	-.10	.02	-.08***	-.03	.02	-.02	-.13	.02	-.10***

***P<0.001 (2-tailed).
**P<0.01 (2-tailed).
*P<0.05 (2-tailed).
[a]men=0, women=1
[b]no=0, yes=1

Table 4
Results from regression analyses testing interactions between family history and other predictors in relation to perceived risk (FINRISK 2007), N=5517−5931.

Interaction term between family history and variable below	CVD B	CVD SE B	CVD *P-value*	Diabetes B	Diabetes SE B	Diabetes *P-value*	Cancer B	Cancer SE B	Cancer *P-value*	Depression B	Depression SE B	Depression *P-value*
Smoking												
Former smokers[a]	-.13	.05	**.004**	-.04	.04	.283	-.01	.03	.691	-.00	.05	.983
Smokers[a]	-.03	.05	.552	.00	.05	.995	-.01	.04	.849	.05	.05	.396
Alcohol	.01	.00	.172	-.00	.00	.571	.00	.00	.412	-.01	.00	**.047**
BMI	-.00	.00	.723	-.00	.00	.420	.00	.00	.522	-.01	.00	**.027**
Physical activity												
Moderately active[b]	.04	.05	.477	.02	.05	.730	-.07	.04	.062	.07	.06	.224
Active[b]	.06	.06	.337	-.00	.06	.980	-.04	.05	.424	.02	.06	.788
Gender	.04	.04	.313	.06	.04	.119	.01	.03	.708	.12	.04	**.005**
Age	-.01	.00	**<.001**	-.01	.00	**.001**	-.01	.00	**<.001**	-.00	.00	.112
Education[c]	.02	.01	**<.001**	.02	.01	**<.001**	.01	.00	.093	.00	.01	.565
Depressive symptoms[d]	-.00	.00	.129	.00	.00	.326	.00	.00	.989			

[a]reference group: never smokers
[b]reference group: sedentary
[c]adjusted for age
[d]subsample N=4513−4605
Interactions between family history and listed variables were tested separately. The interaction term was added to the model after the respective main effects (unadjusted analyses).

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Conflict of Interest Statement

The authors declare that there are no conflicts of interest.

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Highlights

Family history was related to perceived risk of CVD, diabetes, cancer and depression.
The association was strongest for diabetes, weakest for depression.
The associations were independent of sociodemographics and health behavior.
Lay perceptions of heritability need consideration in risk communication practices.